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=> file medline embase biosis caplus  
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FILE 'MEDLINE' ENTERED AT 15:03:07 ON 23 MAY 2006

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=> s skeletal and muscle and damage  
L1 9377 SKELETAL AND MUSCLE AND DAMAGE

=> s l1 and assay  
L2 526 L1 AND ASSAY

=> s l2 and (skeletal(w)troponin(w)I or skeletal(w)myosin(w)light(w)chain or  
skeletal(w)troponin(w)C or skeletal(w)alpha(w)actinin)  
L3 13 L2 AND (SKELETAL(W) TROPONIN(W) I OR SKELETAL(W) MYOSIN(W)  
LIGHT(W) CHAIN OR SKELETAL(W) TROPONIN(W) C OR SKELETAL(W) ALPHA  
(W) ACTININ)

=> dup rem  
ENTER L# LIST OR (END):s3  
S3 IS NOT VALID HERE  
The L-number entered has not been defined in this session, or it  
has been deleted. To see the L-numbers currently defined in this  
session, enter DISPLAY HISTORY at an arrow prompt (=>).

=> dup rem  
ENTER L# LIST OR (END):l3  
PROCESSING COMPLETED FOR L3  
L4 6 DUP REM L3 (7 DUPLICATES REMOVED)

=> dis ibib abs l4

L4 ANSWER 1 OF 6 MEDLINE on STN DUPLICATE 1  
ACCESSION NUMBER: 2005454249 MEDLINE

DOCUMENT NUMBER: PubMed ID: 15833785  
TITLE: Fast and slow **skeletal troponin**  
I in serum from patients with various  
**skeletal muscle** disorders: a pilot study.  
AUTHOR: Simpson Jeremy A; Labugger Ralf; Collier Christine; Brison  
Robert J; Iscoe Steve; Van Eyk Jennifer E  
CORPORATE SOURCE: Department of Physiology, Queen's University, Kingston,  
Ontario, Canada.  
SOURCE: Clinical chemistry, (2005 Jun) Vol. 51, No. 6, pp. 966-72.  
Electronic Publication: 2005-04-15.  
Journal code: 9421549. ISSN: 0009-9147.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200509  
ENTRY DATE: Entered STN: 27 Aug 2005  
Last Updated on STN: 11 Sep 2005  
Entered Medline: 9 Sep 2005

AB BACKGROUND: Detection of **skeletal muscle** injury is hampered by a lack of commercially available **assays** for serum markers specific for **skeletal muscle**; serum concentrations of **skeletal troponin I** (sTnI) could meet this need. Moreover, because sTnI exists in 2 isoforms, slow (ssTnI) and fast (fsTnI), corresponding to slow- and fast-twitch **muscles**, respectively, it could provide insight into differential injury/recovery of specific fiber types. The purpose of this study was to investigate whether the 2 isoforms of sTnI and their modified forms are present in the blood of patients with various **skeletal muscle** disorders. METHODS: Serial serum samples were obtained from 25 patients with various **skeletal muscle** injuries. Serum proteins were separated by a modified sodium dodecyl sulfate-polyacrylamide gel electrophoresis protocol followed by Western blotting for sTnI with monoclonal antibodies specific to ssTnI and fsTnI. RESULTS: We observed (a) intact and, in some cases, degraded sTnI products; (b) evidence of posttranslational modifications in addition to proteolysis; and (c) differential detectability of both **skeletal** isoforms in the same patient. CONCLUSIONS: It is possible to monitor both sTnI isoforms; this could lead to the development of new diagnostic **assays** for **skeletal muscle** damage.

=> dis ibib abs l3 1-6

L3 ANSWER 1 OF 13 MEDLINE on STN  
ACCESSION NUMBER: 2005454249 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 15833785  
TITLE: Fast and slow **skeletal troponin**  
I in serum from patients with various  
**skeletal muscle** disorders: a pilot study.  
AUTHOR: Simpson Jeremy A; Labugger Ralf; Collier Christine; Brison  
Robert J; Iscoe Steve; Van Eyk Jennifer E  
CORPORATE SOURCE: Department of Physiology, Queen's University, Kingston,  
Ontario, Canada.  
SOURCE: Clinical chemistry, (2005 Jun) Vol. 51, No. 6, pp. 966-72.  
Electronic Publication: 2005-04-15.  
Journal code: 9421549. ISSN: 0009-9147.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
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FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200509  
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AB BACKGROUND: Detection of **skeletal muscle** injury is hampered by a lack of commercially available **assays** for serum markers specific for **skeletal muscle**; serum concentrations of **skeletal troponin I** (sTnI) could meet this need. Moreover, because sTnI exists in 2 isoforms, slow (ssTnI) and fast (fsTnI), corresponding to slow- and fast-twitch **muscles**, respectively, it could provide insight into differential injury/recovery of specific fiber types. The purpose of this study was to investigate whether the 2 isoforms of sTnI and their modified forms are present in the blood of patients with various **skeletal muscle** disorders. METHODS: Serial serum samples were obtained from 25 patients with various **skeletal muscle** injuries. Serum proteins were separated by a modified sodium dodecyl sulfate-polyacrylamide gel electrophoresis protocol followed by Western blotting for sTnI with monoclonal antibodies specific to ssTnI and fsTnI. RESULTS: We observed (a) intact and, in some cases, degraded sTnI products; (b) evidence of posttranslational modifications in addition to proteolysis; and (c) differential detectability of both **skeletal** isoforms in the same patient. CONCLUSIONS: It is possible to monitor both sTnI isoforms; this could lead to the development of new diagnostic **assays** for **skeletal muscle** damage.

L3 ANSWER 2 OF 13 MEDLINE on STN  
ACCESSION NUMBER: 96426681 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 8828960  
TITLE: Use of enzyme immunoassay for measurement of **skeletal troponin-I** utilizing isoform-specific monoclonal antibodies.  
AUTHOR: Takahashi M; Lee L; Shi Q; Gawad Y; Jackowski G  
CORPORATE SOURCE: Spectral Diagnostics, Inc., Toronto, Ontario, Canada.  
SOURCE: Clinical biochemistry, (1996 Aug) Vol. 29, No. 4, pp. 301-8.  
Journal code: 0133660. ISSN: 0009-9120.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199702  
ENTRY DATE: Entered STN: 27 Feb 1997  
Last Updated on STN: 27 Feb 1997  
Entered Medline: 11 Feb 1997

AB OBJECTIVE: To determine the serum level of fast **skeletal troponin I** (fsTnI) resulting from **skeletal muscle damage**, we have developed a sensitive two-site enzyme immunoassay to measure **skeletal troponin I**. DESIGN AND METHODS: Twelve monoclonal antibodies were raised against human fsTnI. Of these antibodies, 8 were fsTnI-specific and the remaining 4 reacted with both **skeletal** and cardiac troponin I (cTnI). Two monoclonals were utilized for a development of this fsTnI immunoassay. Standards were made with purified recombinant human fsTnI for the range of 0-25 micrograms/mL. RESULTS: Total **assay** variance (CV) ranged from 1.7% to 9.6%. The upper limit of the normal reference range was established as 0.2 microgram/L by determining fsTnI concentration in sera of 108 healthy donors without evidence of **muscle damage**. Purified human cTnI up to 500 micrograms/L and cTnI-positive clinical serum samples yielded negative results in the fsTnI **assay**. The serum levels of fsTnI were determined in trauma patients, patients with chronic degenerative **muscle** disease, and marathon runners. In the study populations, the serum levels of fsTnI were correlated with other biochemical markers that are traditionally used to monitor striated **muscle damage**. CONCLUSIONS: In the present preliminary studies, measuring the serum levels of fsTnI in patients with various forms of **muscle damage** is more accurate than using the classical non **muscle**-specific biochemical markers.

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ACCESSION NUMBER: 2005240815 EMBASE  
TITLE: Fast and slow **skeletal troponin I** in serum from patients with various **skeletal muscle disorders**: A pilot study.  
AUTHOR: Simpson J.A.; Labugger R.; Collier C.; Brison R.J.; Iscoe S.; Van Eyk J.E.  
CORPORATE SOURCE: S. Iscoe, Department of Physiology, Queen's University, Kingston, Ont. K7L 3N6, Canada. iscoes@post.queensu.ca  
SOURCE: Clinical Chemistry, (2005) Vol. 51, No. 6, pp. 966-972. .  
Refs: 28  
ISSN: 0009-9147 CODEN: CLCHAU  
COUNTRY: United States  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 005 General Pathology and Pathological Anatomy  
029 Clinical Biochemistry  
033 Orthopedic Surgery  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 30 Jun 2005  
Last Updated on STN: 30 Jun 2005

AB Background: Detection of **skeletal muscle injury** is hampered by a lack of commercially available **assays** for serum markers specific for **skeletal muscle**; serum concentrations of **skeletal troponin I (sTnI)** could meet this need. Moreover, because sTnI exists in 2 isoforms, slow (ssTnI) and fast (fsTnI), corresponding to slow- and fast-twitch **muscles**, respectively, it could provide insight into differential injury/recovery of specific fiber types. The purpose of this study was to investigate whether the 2 isoforms of sTnI and their modified forms are present in the blood of patients with various **skeletal muscle disorders**. Methods: Serial serum samples were obtained from 25 patients with various **skeletal muscle injuries**. Serum proteins were separated by a modified sodium dodecyl sulfate-polyacrylamide gel electrophoresis protocol followed by Western blotting for sTnI with monoclonal antibodies specific to ssTnI and fsTnI. Results: We observed (a) intact and, in some cases, degraded sTnI products; (b) evidence of posttranslational modifications in addition to proteolysis; and (c) differential detectability of both **skeletal isoforms** in the same patient. Conclusions: It is possible to monitor both sTnI isoforms; this could lead to the development of new diagnostic **assays for skeletal muscle damage**.  
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ACCESSION NUMBER: 2004133429 EMBASE  
TITLE: [Evaluation of a rapid immunoassay for the quantification of cardiac troponin I in the diagnosis of acute myocardial infarction].  
EVALUACION DE UN INMUNOANALISIS RAPIDO DE CUANTIFICACION DE TROPONINA I CARDIACA EN EL DIAGNOSTICO DE INFARTO AGUDO DEL MIOCARDIO.  
AUTHOR: Mainet Gonzalez D.; Sorell Gomez L.; Pichardo Diaz D.; Reyes Acosta O.; Torres Cabrera M.B.; Abdo Cuza A.; Castellano Gutierrez R.; Padron Brito N.  
CORPORATE SOURCE: D. Mainet Gonzalez, Ctro. Ing. Genet./Biotecnol., Apartado postal 6162, Ciudad Habana 10600, Cuba.  
damian.mainet@cigb.edu.cu  
SOURCE: Quimica Clinica, (2003) Vol. 22, No. 6, pp. 419-430. .  
Refs: 49  
ISSN: 1139-2436 CODEN: RSQCFW  
COUNTRY: Spain

DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 005 General Pathology and Pathological Anatomy  
018 Cardiovascular Diseases and Cardiovascular Surgery  
LANGUAGE: Spanish  
SUMMARY LANGUAGE: English; Spanish  
ENTRY DATE: Entered STN: 12 Apr 2004  
Last Updated on STN: 12 Apr 2004

AB Cardiac troponin I is considered the biochemical marker of choice in acute myocardial infarction due to its high cardioespecificity. Twenty one monoclonal antibodies were obtained that recognized several epitopes of the cardiac troponin I in the free form and forming complexes with troponin T and troponin C. We were able to standardize an immunoassay with a duration of less than one hour for the quantification of cardiac troponin I in plasma. The following parameters of the immunoassay were evaluated: inter-assay and intra-assay coefficients were smaller than 10%, limit of detection of 0.1 µg/L, (as percent recovery) accuracy between 90% - 110% and absence of cross reactivity with the skeletal troponin I. The clinical specificity was 100% in the retrospective evaluation of this biochemical marker in healthy donors, patients with unstable angina, patients with chronic renal failure and patients with skeletal muscle damage. The clinical sensitivity was 100% from 24 to 48 hours and 98,2% from 6 to 48 hours from the onset of chest pain in patients with acute myocardial infarction. For these reasons, this immunoassay fulfills the recommendations suggested by the international committee of standardization of this biochemical marker for the use in coronary cares units.

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ACCESSION NUMBER: 97300602 EMBASE  
DOCUMENT NUMBER: 1997300602  
TITLE: Analytical performance and clinical utility of a sensitive immunoassay for determination of human cardiac troponin I.  
AUTHOR: Davies E.; Gawad Y.; Takahashi M.; Shi Q.; Lam P.; Styba G.; Lau A.; Heesch C.; Usategui M.; Jackowski G.  
CORPORATE SOURCE: E. Davies, Spectral Diagnostics Inc., 135-2 West Mall, Toronto, Ont. M9C 1C2, Canada  
SOURCE: Clinical Biochemistry, (1997) Vol. 30, No. 6, pp. 479-490.

Refs: 28  
ISSN: 0009-9120 CODEN: CLBIAS

PUBLISHER IDENT.: S 0009-9120(97)00111-2  
COUNTRY: United States  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 029 Clinical Biochemistry  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 16 Oct 1997  
Last Updated on STN: 16 Oct 1997

AB Objectives: To determine the serum and plasma level of human cardiac troponin I (cTnI) resulting from myocardial damage, we have developed a sensitive and specific one-step enzyme immunoassay to measure cardiac troponin I. Design and Methods: The COBAS® cTnI assay is a semi-automated one-step solid phase immunoassay compatible with the COBAS® Core. The assay is performed in a sandwich type format using a polyclonal goat antibody capture and two highly specific horseradish peroxidase conjugated monoclonal antibody detectors directed against different epitopes of the cTnI molecule. Calibrators were made with purified recombinant cTnI. Results: The level of cTnI was determined in 84 healthy donors with no evidence of myocardial injury, resulting in a lower limit of detection (LLD) of 0.09 µg/L. The upper reference limit (URL) of the normal reference range was calculated as 0.20 µg/L. The dynamic range of the consequent EIA was between 0.09 and 6.0 µg/L with a total assay time of 45 min. Intra-assay and inter-

assay variances (CVs) were  $\leq 4\%$ . Cross-reactivity with fast and slow **skeletal troponin I** was absent in concentrations up to 2.0 mg/L. Common interferents yielded negative results in the cTnI assay. Clinical utility was confirmed by measuring the circulating serum or plasma levels of cardiac troponin I in serial samples from marathon runners, clinical samples from trauma patients, and patients presenting to the Emergency Department with complaints of chest pain. Results were further evaluated using clinical diagnosis at discharge and quantified concentrations of other cardiac markers by a Stratus® analyzer and ELISA procedures. Conclusions: Results from normal and clinical samples assayed in-house for cTnI concentrations indicate that the Spectral EIA is a highly sensitive means of quantifying cTnI levels in serum and plasma for acute cardiac syndrome. The cardiac specificity of cTnI over other well-known cardiac markers is reflected in experimental results and parallel clinical diagnosis.

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ACCESSION NUMBER: 96227677 EMBASE

DOCUMENT NUMBER: 1996227677

TITLE: Use of enzyme immunoassay for measurement of **skeletal troponin-I** utilizing isoform-specific monoclonal antibodies.

AUTHOR: Takahashi M.; Lee L.; Shi Q.; Gawad Y.; Jackowski G.

CORPORATE SOURCE: Spectral Diagnostics, Inc., 135 West Mall, Toronto, Ont. M9C 1C2, Canada

SOURCE: Clinical Biochemistry, (1996) Vol. 29, No. 4, pp. 301-308.

ISSN: 0009-9120 CODEN: CLBIAS

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 008 Neurology and Neurosurgery  
029 Clinical Biochemistry

*August*

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 7 Oct 1996

Last Updated on STN: 7 Oct 1996

AB Objectives: To determine the serum level of fast **skeletal troponin I** (fsTnI) resulting from **skeletal muscle damage**, we have developed a sensitive two-site enzyme immunoassay to measure **skeletal troponin I**. Design and Methods: Twelve monoclonal antibodies were raised against human fsTni. Of these antibodies, 8 were fsTnI-specific and the remaining 4 reacted with both **skeletal** and cardiac troponin I (cTnI). Two monoclonals were utilized for a development of this fsTnI immunoassay. Standards were made with purified recombinant human fsTnI for the range of 0-25  $\mu\text{g/mL}$ . Results: Total assay variance (CV) ranged from 1.7% to 9.6%. The upper limit of the normal reference range was established as 0.2  $\mu\text{g/L}$  by determining fsTnI concentration in sera of 108 healthy donors without evidence of **muscle damage**. Purified human cTnI up to 500  $\mu\text{g/L}$  and cTnI-positive clinical serum samples yielded negative results in the fsTnI assay. The serum levels of fsTnI were determined in trauma patients, patients with chronic degenerative **muscle** disease, and marathon runners. In the study populations, the serum levels of fsTnI were correlated with other biochemical markers that are traditionally used to monitor striated **muscle damage**. Conclusions: In the present preliminary studies, measuring the serum levels of fsTnI in patients with various forms of **muscle damage** is more accurate than using the classical non **muscle**-specific biochemical markers.

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NEWS	15	APR 12	Derwent World Patents Index to be reloaded and enhanced during second quarter; strategies may be affected
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NEWS	18	MAY 19	Derwent World Patents Index to be reloaded and enhanced
NEWS EXPRESS			FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005. V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT <a href="http://download.cas.org/express/v8.0-Discover/">http://download.cas.org/express/v8.0-Discover/</a>
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COST IN U.S. DOLLARS

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ENTRY	SESSION
1.26	52.65

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